OSHA: Proposed Standard For Indoor Air Quality: ETS Hearings, January 17, 1995

UNITED STATES DEPARTMENT OF LABOR

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

PUBLIC HEARING
PROPOSED STANDARD FOR INDOOR AIR QUALITY

Tuesday,
January 17, 1995

Department of Labor

Washington, D.C.

The above-entitled matter came on for hearing, pursuant to notice, at 9:45 a.m.

BEFORE: HONORABLE JOHN VITTONE

Administrative Law Judge

AGENDA

PAGE

Questions:

Ms. Sherman 11543

EXHIBITS

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P R O C E E D I N G S
9:45 a.m.

JUDGE VITTONE: Good morning, ladies and gentlemen. We resume our hearings into the proposed rule by the Occupational Safety and Health Administration on indoor air quality.

For the record, this is day 51 of these hearings.

We have on this morning a panel from the R.J. Reynolds Tobacco Company that will be testifying today and will be also here for the next several days, Tuesday, Wednesday and Thursday of this week.

I would like to begin by introducing the chairman of the RJR panel, Dr. Coggins.

DR. COGGINS: Thank you, Judge Vittone.

Good morning, ladies and gentlemen. I am Chris Coggins. I hold a Ph.D. from the University of Edinburgh, Scotland, and I am a board certified toxicologist.

JUDGE VITTONE: Excuse me a second. I'm sorry. I forgot something here. I have a preliminary matter that somebody wanted to raise with me.

Mr. McNeely, would you come forward, please?

MR. McNEELY: Actually, Mr. Herman will be making --

JUDGE VITTONE: Okay. Mr. Herman.

Why don't you come on around here, Mr. Herman?

MR. HERMAN: Judge Vittone, I am not at all certain of the procedure that will be followed this morning.

JUDGE VITTONE: I will be glad to explain it to you, then. What we plan to do here today, and I guess I should advise everyone here, sometimes I tend to forget that not everybody understands how we operate, what we will be doing over the next several days is simply this: the R.J. Reynolds panel will be making a presentation today, individuals members of the panel will be called at various times and make a presentation on various subject matter, on topics, I'm not sure exactly what each person will be talking about.

We will be taking them in seriatim. Dr. Coggins is the chairman, as I understand, of the panel and then he will call upon individual members of this panel to speak on various topics.

That will probably consume all of this morning and at least a portion of this afternoon's proceeding. After they have completed their direct testimony, using the slides and everything, then they will be made available for examination by the participants in the proceeding.

I will then turn to Ms. Sherman, who heads up the OSHA staff. Ms. Sherman and her staff will be able to examine the panel about their testimony. I do not know how long that will be but I am sure it it's gong to be lengthy examination.

After she has completed her direct examination, then I will make the panel available for examination by other participants in this proceeding who have complied with the rules with respect to becoming actual parties or participants to the proceeding and that will probably take us through some time on Thursday.

Then at that point after all of the participants have completed their examination, if there is any brief clarification that the panel members want to make, I will give them an opportunity to make a statement at that point and then we will end with them.

It is the same pattern we have pretty much followed in this proceeding for every other witness or panel of witnesses that have testified in this proceeding so far, including the NIOSH panel, the American Medical Association, the unions that have testified, anybody else.

So that's basically our regular process, so that's it.

MR. HERMAN: May I ask if any of these gentlemen have engagements that will cause them to leave at any time before we have an opportunity to fully speak with them about the issues they have raised?

JUDGE VITTONE: Well, I think we've gone over this but I'll give Mr. Grossman an opportunity to respond to that.

Mr. Grossman, since you haven't been back for a while, why don't you tell everybody who you are?

MR. GROSSMAN: I will. Your Honor, my name is Ted Grossman. I am here on behalf of R.J. Reynolds.

The panel, all of the members of the panel, have reserved today and tomorrow and Thursday for these hearings as the schedule calls for. Obviously they haven't scheduled an indefinite appearance and neither has any other organization.

JUDGE VITTONE: So they will be here.

Now, just so we are clear, the OSHA panel has also scheduled for Thursday two other individuals which we will bring on at the completion of the RJR panel, Mr. Allan Hedge and Mr. Gene Davidson. I don't know anything about those gentlemen but they will be here on Thursday but they will be here after the RJR panel is completed.

MR. GROSSMAN: Yes, Your Honor. We scheduled them in accordance with the schedule, has OSHA has.

JUDGE VITTONE: Okay.

Mr. Herman, does that answer your question, sir?

MR. HERMAN: Yes, it does. And I understand that thankfully we will have a full opportunity because the entire panel will be here for the duration.

MR. GROSSMAN: For the duration of Reynolds' testimony. Yes.

MR. HERMAN: And cross.

MR. GROSSMAN: Yes.

MR. HERMAN: Your Honor, I have only one comment.

JUDGE VITTONE: Okay.

MR. HERMAN: And I appreciate your allowing me to take this time just to clarify what the procedure was.

We have an objection in principal to allowing RJR to appear en masse like this. It really thwarts the ability, our ability, to examine each of the gentlemen individually and it allows RJR to take the stage for a very long period of time without breaking the continuity and allows them in effect to have a continuing commercial for the benefit of the cigarette industry.

We understand your ruling and we appreciate it and our concern is that while unintentional certainly the way that this process is being allowed falls square into the concerns that those of us who represent the other side of this case are most concerned about and that is a full and complete airing of the issues without the opportunity, though we respect your ruling, I want to state it for the record, without the opportunity of being able to question each of these gentlemen after their testimony is concluded. It's going to adversely affect our ability to get a full and complete airing of our side of the issue.

And having said that, I appreciate the time you have allowed me.

JUDGE VITTONE: You are welcome, sir.

Mr. Grossman?

MR. GROSSMAN: Thank you, Your Honor. If I may respond briefly, we've been across this bridge many times before, both as to Reynolds and as to others, and, as Your Honor has already said, every other panel, the AMA, OSHA, NIOSH, has testified in precisely this way. The only difference, I might add, is that Reynolds has identified the authors of the submissions that it has before you.

Mr. McNeely raised exactly this point on December 7th and at page 8686 of the transcript --

JUDGE VITTONE: Mr. Grossman, I don't want to cut you off, I'll give you an opportunity to respond but I do not intend to change my ruling in this matter.

MR. GROSSMAN: I was just going to say at that page you said that there would be no revision and no reconsideration of your ruling.

JUDGE VITTONE: My ruling stands. I will permit the RJR panel to proceed, present its entire direct testimony and then be made available for examination.

Thank you, Mr. Herman, for your comments. I understand your position and it's the same position that the OSHA staff took but it's a position I decided not to adopt and decided to proceed in this manner and this is the way we will go forward.

Thank you, Mr. Herman.

Thank you, Mr. Grossman.

Anything else before we begin?

(No audible response)

JUDGE VITTONE: Seeing no other hands raised, Dr. Coggins, you can resume what you were saying before.

DR. COGGINS: Thank you, Judge Vittone.

Good morning, ladies and gentlemen. I am Chris Coggins. I hold a Ph.D. from the University of Edinburgh in Scotland and I am a board certified toxicologist. I am a Principal Scientist in the Research and Development Department of the R.J. Reynolds Tobacco Company. I have performed and published toxicological evaluations with tobacco smoke since I received my doctorate in 1976.

My colleagues and I are here today to present data that will illustrate a number of serious shortcomings in OSHA's proposed rule as it relates to environmental tobacco smoke, ETS.

We hope that our information will help OSHA develop a better, more appropriate approach to ETS in the workplace.

As you will hear, Reynolds Tobacco believes that the proposed rule requires substantial modification. In particular, we do not believe there is any justification for treating ETS in a different manner from other indoor air components.

When we have completed our presentations, we will answer questions concerning them. I will be the moderator for the panel and I will direct the questions to the most appropriate panel member.

Now, let me introduce our panel.

Michael Ogden, who received his Ph.D. in analytical chemistry from Virginia Tech in 1985, will speak first. Dr. Ogden will present data on the actual amounts of ETS that are found in a number of different environments, including workplaces and homes. Dr. Ogden will show that non-smoker exposures to ETS are typically very much smaller than those cited in the proposed rule.

Next, Paul Nelson, who obtained his Ph.D. in analytical chemistry from Georgia Tech in 1987, will talk about the severe limitations in the use of nicotine and cotinine to precisely predict ETS exposure.

Then, Stephen Sears, on my left, who obtained his Ph.D. in theoretical chemistry from UNC at Chapel Hill in 1980, will review some of the problems in the OSHA risk assessments, problems that in the main have not yet been raised at these hearings.

Dr. Sears' presentation was co-written by Mr. Thomas Steichen, who obtained his Master's degree in statistics from the University of Kentucky in 1978. Mr. Steichen will also be on our panel to answer questions.

After that, Dr. Ogden will return to present data showing that misclassification affects the results in epidemiologic studies concerning ETS.

I will then analyze some animal inhalation studies. I will show that the studies used in the proposed rule are inappropriate and that more relevant studies were not given the attention they deserve.

Finally, Mr. Hoy Bohanon, who obtained his Bachelor's degree in mechanical engineering from Georgia Tech in 1974, will discuss engineering solutions as alternatives to a national smoking ban. Mr. Bohanon is a professional engineer.

Our presentations will demonstrate why from a scientific and technical point of view there is no justification for attempting to impose a zero exposure standard for ETS in the workplace and why proper ventilation under the general IAQ standard is the least costly, least intrusive and best way to deal with workplace ETS issues.

Now, Dr. Ogden will present data on the actual amounts of ETS that are found in a number of different environments, including workplaces and homes.

Dr. Ogden will be using the slide projector.

JUDGE VITTONE: Okay. Each of you has slides, as I understand it, which you will be using.

DR. COGGINS: That's correct.

JUDGE VITTONE: I have been provided with copies of your presentation as well as copies of the slides.

With the completion of each witness I will identify by exhibit number his presentation and slides for the record.

It is also my understanding that there are copies of your statements and slides available in the back, outside the hall here?

DR. COGGINS: That's correct.

JUDGE VITTONE: Okay. So they are on the table back there for anybody who may want to see them.

DR. COGGINS: Correct.

JUDGE VITTONE: Thank you very much.

We will proceed with Dr. Ogden if you are ready, sir.

Are you going to be using the slides right away, Doctor?

DR. OGDEN: Yes.

JUDGE VITTONE: Let me move out of your way.

(Pause)

DR. OGDEN: Thank you, Dr. Coggins, Your Honor.

Good morning. I am Dr. Michael Ogden. I hold a Bachelor of Science degrees in both chemistry and applied mathematics and a Ph.D. in analytical chemistry.

Since 1985, I have been employed by R.J. Reynolds Tobacco Company in the Research and Development Department. For the past nine years, my research has been almost exclusively focused on studying environmental tobacco smoke by developing and applying methods for assessing ETS exposure.

My testimony will address the treatment of exposure to ETS in the Notice of Proposed Rulemaking and comments made by OSHA consultants.

In slide 1, I pose five questions. They are:

(1) How should we measure exposure to ETS?

(2) What is the correct definition of exposure?

(3) How much ETS is actually in the workplace?

(4) How does ETS exposure compare for living with smokers and working with smokers?

(5) How many workers are actually exposed in their workplace?

Significant new data relevant to each of these questions and, more specifically, to OSHA's assertions are also summarized and introduced at the appropriate places.

Before OSHA can regulate any workplace exposure, including ETS, it must demonstrate with substantial evidence that a significant risk of material impairment to health is present in the workplace.

In addition, OSHA has to know the answer to two very basic questions and these are how much ETS is in the air and how many workers are exposed. The NPR does not adequately answer either of these questions. As a result, the proposed workplace ETS rule is based on inadequate information.

In my testimony here today, I will demonstrate the following points as outlined in slide 2.

(1) Methodology for measuring exposure. OSHA has not measured ETS exposure in the workplace and has not considered the most up-to-date and relevant information on how to go about measuring the exposure.

(2) The definition of exposure. In much of the NPR, OSHA is apparently using an incorrect definition of exposure.

(3) The concentration of ETS in the workplace. OSHA doesn't know how much ETS is in the typical workplace. As I will demonstrate, typical exposure levels are up to 250 times lower than assumed by OSHA.

(4) Equating home and workplace exposures. In lieu of actually measuring exposure, OSHA attempts to equate workplace exposure for people who work with smokers with home exposure for people who live with smokers. Research shows that instead of these exposures being equal, the workplace exposures are actually five to 10 times lower than home exposures.

(5) The number of workers exposed. OSHA doesn't know how many workers are actually exposed to ETS at work. They have significantly overestimated the number of workers exposed.

The important point is this: Without the best available answers to any of these questions, there is only one logical outcome: OSHA's conclusions regarding ETS in the NPR are not meaningful.

I'll now turn to the first question I posed: How do we actually measure exposure to ETS? Quite simply, we can measure people's exposure to chemicals in the air that are attributable to ETS. These compounds are called markers. This is slide 3.

In May 1993, I presented to OSHA a summary of the commonly used ETS exposure markers. In that presentation, I concluded that two markers, 3-ethenylpyridine and solanesol, were the best available markers for the vapor phase and the particulate phase, respectively, of ETS.

Nicotine is a commonly used marker for ETS; however, it is inferior to both 3-ethenylpyridine and solanesol, particularly at low concentrations.

Recently, these conclusions regarding nicotine and 3-ethenylpyridine have been questioned by Dr. Katherine Hammond at these hearings. I would like to address her criticisms here but before I do I

would like to present some additional background information that is relevant to support my qualifications to make these conclusions.

I developed and published the method for measuring nicotine in ETS that is referred to as the XAD-4 nicotine method. As outlined here in slide 4, this method is currently the most thoroughly tested, the most rigorously validated, and the most widely used method in the world.

I currently serve as Association of Official Analytical Chemists Associate Referee for studying nicotine in environmental tobacco smoke. This title is used to designate a technical expert charged with supervising method validation and providing statistical analysis of the results and writing the technical protocol.

This XAD-4 nicotine method has undergone two successful international collaborative studies. As shown in slide 5, it is an approved or official method of the following organizations: The Association of Official Analytical Chemists, the U.S. Environmental Protection Agency, and the American Society for Testing and Materials. Also, this method is currently a draft international standard within ISO, the International Standards Organization.

It is currently in use by researchers in at least eight countries. The analysis is also available in at least four commercial laboratories in the U.S. and one in Canada. This same method is also used for the determination of 3-ethenylpyridine in ETS.

Testifying on behalf of OSHA, Dr. Hammond correctly concluded that one of the most important attributes of a marker is that its concentration should increase with the source strength and reflect the concentration of the complex mixture, here, ETS. However, she incorrectly concluded that 3-ethenylpyridine fails this most fundamental test for a marker.

In her Figure 1, which I have reproduced here in slide 6, Dr. Hammond shows results from one experiment done by another researcher in which both nicotine and RSP increase more or less linearly with the number of cigarettes smoked. However, the data for 3-ethenylpyridine do not appear to increase in the same fashion. Not only is this result for 3-ethenylpyridine implausible, it is incorrect.

Shown here in slide 7 are the ETS vapor phase marker results from a similar experiment conducted at R.J. Reynolds. These data demonstrate two points.

First, 3-ethenylpyridine increases linearly with the number of cigarettes smoked. Second, 3-ethenylpyridine also tracks exactly the vapor phase of ETS, as measured by carbon monoxide and flame ionization detector response, which is an indication of the total volatile organic compounds present in ETS.

As you can see, although nicotine increases in a similar fashion, it actually overestimates the vapor phase of ETS. This overestimation becomes more predominant with increasing levels of smoke.

Myosmine also exhibits the same trend as nicotine, although to a lesser extent.

Dr. Hammond levies an additional criticism against 3-ethenylpyridine. She claims that 3-ethenylpyridine is not as sensitive a marker as nicotine in detecting ETS. This also is not true.

In 1992, we published limits of detection for both nicotine and 3-ethenylpyridine showing that the that 3-ethenylpyridine is actually twice as easy to detect as nicotine.

Slide 8 shows the ETS particulate phase marker results from the same experiment at RJR. As for the vapor phase components, all markers increase linearly with increasing concentration. However, all particulate phase markers track each other very well. This is to be expected in this controlled experiment where all RSP comes from ETS.

In addressing how to go about measuring ETS exposure, the NPR concludes that the use of an internal measure of individual exposure such as body fluid cotinine is preferable to actually measuring external exposure. I disagree with this statement.

While an internal measure may be well suited to some experiments in the workplace, it is certainly not true with regard to ETS.

First of all, of the potential biomarkers currently available for estimating exposure to ETS, cotinine is the only one that is even marginally useful.

Of all biomarkers that have been proposed, cotinine is the most tobacco-specific. Also, cotinine has a half-life of approximately 17 hours, although it certainly can vary over a much wider range. This was discussed in more detail previously by Dr. Neil Benowitz.

This half-life is the amount of time it takes for half of any nicotine inhaled to be eliminated from the body as cotinine. This means that at best cotinine provides an integrated estimate of exposure over the preceding one to three days.

However, in the context of measuring workplace ETS exposure, cotinine is not to be preferred over air monitoring. There are several reasons for this, however, I will explain only one of them here in slide 9.

A significant problem with the use of cotinine for workplace exposure assessment is this same relatively long half-life that I just mentioned. By relatively long half-life, I now mean relative to the continuous amount of time spent at work.

The continuous amount of time spent in the workplace for most workers is only about eight hours. This continuous amount of time would even be less for workers who may leave their workspace to run errands, go out to eat, et cetera.

In order to use cotinine or any other biomarker with a similar half-life to infer anything about exposure at work requires the implicit assumption that all out-of-workplace exposures are the same for everyone.

This is, of course, an illogical assumption. An individual's body fluid levels of cotinine cannot distinguish between nicotine inhaled at work, at lunch, at home or anywhere else.

I would like to cite one specific example from my own research that illustrates how relying solely on cotinine levels would have resulted in a serious error concerning potential workplace exposures.

This study, outlined in slide 10, was conducted in Columbus, Ohio in 1991. The results were presented to OSHA in May 1993, and were published later that year.

Among all non-smoking subjects who were exposed to ETS at home, a statistically significant

difference in cotinine levels was found between those who worked outside the home and those who did not work outside the home. In short, the subjects who were spousal-exposed to ETS at home and who also worked outside the home had higher cotinine levels than subjects who were spousally exposed at home and who did not work outside the home.

If I had relied solely on cotinine data, I would have attributed this increased cotinine to ETS exposure at work. However, I would have been wrong.

Air monitoring in the homes revealed higher concentrations of nicotine and 3-ethenylpyridine in the home of the working subject. Thus, the difference in cotinine was truly attributable to a difference in home exposure and was not due to a workplace effect.

Personal monitoring of workers going about their daily activities in their workplace is a much better way to determine actual exposure.

I would like to offer this specific advice to OSHA: If you want to know what exposure is in the workplace, measure it.

With readily available materials and methods, a number of informative constituents of ETS can be measured, including 3-ethenylpyridine, solanesol and even nicotine.

From the quote in slide 11, you can see that Meridian Research reached the same conclusion regarding ETS exposure in the workplace. This was in a 1988 report which was commissioned by OSHA.

Moving to my next point in slide 12, now let's examine the proper definition of exposure and contrast that to what was actually used in the NPR.

Since it is critical in evaluating the merits of this entire section of the NPR, the correct definition of exposure must first be given.

Exposure can correctly be defined simply as being equal to concentration times time. This equation defining exposure is explicit in that exposure in any given environment is equally dependent upon both the time spent in that environment and the concentration level of the contaminant in that environment. Without assessment of both variables, exposure cannot be determined.

Let me illustrate with a simple analogy. Remember that exposure is equally dependent on two variables, time and concentration. Similarly, the distance you drive in your car is dependent on two variables, the amount of time you spend in your car and the average speed of the car.

Imagine if you will trying to estimate how far I drove yesterday if all I tell you is that I spent an hour and a half in my car.

If I were at home in North Carolina yesterday in the middle of the afternoon, I could have driven all the way from Winston-Salem to Durham, a distance of about 75 miles. If I were here in Washington yesterday during rush hour, I might not have made it the four miles or so to the Key Bridge.

The treatment of ETS exposure among the non-smoking, working U.S. population in the NPR is superficial and extremely problematic. In fact, OSHA consistently uses an incorrect definition of exposure in the NPR in sections which focus only on duration.

OSHA employs comparisons of reported durations of ETS exposure between homes and workplaces where smoking occurs.

The data sources on which OSHA relies, such as the CAP survey, only yield estimates of potential exposure. The vast majority of information cited in this section of the NPR contains no measurements of workplace exposure whatsoever, an omission that most seriously limits the development of a workplace risk assessment.

Compounding the problem of using an incorrect definition, OSHA also has improperly analyzed many of the studies on which it does rely.

For example, in the NPR's treatment of the California Activity Pattern, or CAP, survey, this contains a number of errors and misinterpretations. I'll cite just one example from the CAP survey.

The NPR claims the CAP study shows that 51 percent of male and 38 percent of female non-smokers reported ETS exposure at work and further claims that this verifies the high percentage of non-smokers who are exposed to ETS while at work.

These figures actually represent the percentages of exposure time reported to occur at work for non-smokers who reported exposure to ETS at any location. These are not the percentages of the working populations exposed, as stated in the NPR.

Additional shortcomings of the CAP survey and OSHA's interpretation of it in the NPR are detailed in my written submission.

Similar problems occur in two other studies cited predominantly in the NPR, the studies of Cummings et al. and Emmons et al. In these two studies, the authors actually incorporated an analytical measure of ETS exposure in addition to subjective responses regarding potential exposure duration.

The NPR failed to cite the analytical data from either study and chose instead to rely entirely on potential exposure duration as an inappropriate surrogate for exposure. Additional details on these two studies will be provided later.

As I stated earlier, OSHA is obligated to determine how much smoke is actually in the workplace air before they can move to regulate it.

Moving now to my third point, let's see how OSHA went about determining this critical information.

In an attempt to establish the concentration of ETS in the workplace, OSHA has relied on a limited number of outdated, non-representative and extreme data sets. Proper consideration of recent, representative personal monitoring studies demonstrates that the NPR has overestimated typical worker exposures at least 10 to 100 times.

In the NPR, four studies are cited to support the conclusion that the average RSP level during smoking in smoking buildings was 262 micrograms per cubic meter while in non-smoking buildings the RSP levels averaged 36 micrograms per cubic meter. This is slide 13.

Of the four citations given in support, one is obviously an incorrect citation and cannot be verified. The other three include a report by First and two from Repace and Lowery. These three studies describe RSP measurements made with a portable piezobalance over a decade ago.

There are four reasons why this is important. These are outlined in slide 14.

First, without getting into too much technical detail about this measurement device, suffice it to say that the portable piezobalance is most often used for short-term measurements. This is because it requires substantial maintenance between measurements to keep it operating adequately.

Accordingly, the levels reported are in general short-term, peak concentrations, not long-term, time averaged concentrations, the latter of which is needed to truly characterize exposure. For example, Repace and Lower report most sampling times are only 10 to 20 minutes.

Second, the suitability of this type of measurement device has been questioned by First, one of the authors cited for the measurements. In his opinion, First says, and I quote, "This apparatus was not designed for this type of service and lacks the sensitivity and precision needed to sense the small incremental concentrations attributable to tobacco smoking in public places."

Third, the portable piezobalance is meant to measure area concentrations, not personal exposures of people going about their normal activities.

And, fourth, it is readily apparent that typical smoking behavior in public in the 1990s is much different from smoking behavior when these measurements were taken in the late 1970s and early 1980s.

While measurements of the magnitude reported in these studies are possible, they are far from being typical, at least in the 1990s. These extreme values need to be viewed in the context in which they were generated and also compared to modern day, realistic RSP concentrations in workplaces with and without smokers.

In contrast to these area monitoring values cited in the NPR, I presented in my written submission the personal monitoring results from two recent, representative, population-based surveys of workplace RSP levels. These data are summarized here in slide 15.

One of these is a 24-hour personal monitoring study which we conducted in Mt. Laurel, New Jersey in 1992. A detailed report of this study is included as an appendix to my written submission. The other, a recently completed nationwide 24-hour personal monitoring study being conducted by Oak Ridge National Laboratory is also described in a report by Jenkins et al. to the docket.

In both of these studies, total RSP is measured in both smoking and non-smoking workplaces. In addition, and more importantly, both studies also include direct measurements of ETS RSP or the amount of RSP attributable to ETS based on solanesol determination. The exact details of the method used, sometimes referred to as Sol-PM, are described in my written submission and in the literature.

The results of these two studies are shown in slide 15, along with the values cited in the NPR.

These results show the total RSP levels in the average smoking workplace are at least five times lower than stated in the NPR. More important, and more relevant, the largest and most recent data set of Jenkins et al. also shows that ETS RSP levels are over 200 times lower than assumed in the NPR.

In summarizing workplace data for RSP, the NPR concludes that RSP is elevated 10 to 100 times during smoking. There are some extreme data in the literature that can be used to support this

contention. However, as slide 16 correctly summarizes, recent data from truly representative studies show that total RSP is elevated only by factors of two to three, not 10 to 100, and, of course, only a fraction of this RSP is attributable to ETS.

OSHA's summary statement at best represents short-term, peak concentrations in selected environments measured more than 10 years ago and does not reflect the current status of long-term exposures in workplaces in the U.S. that permit smoking.

These typical increases in RSP levels of only two to three times in workplaces that permit smoking are also supported by two additional studies which are cited in the NPR. These data are summarized in slide 17.

In a study by Spengler et al., personal RSP exposure levels differed by less than a factor of three, 34 versus 13 micrograms per cubic meter between exposed and non-exposed groups.

In a study by Sexton et al. for workplace exposures, reported times in excess of two hours per day of ETS exposure resulted in a personal RSP level of 39 micrograms per cubic meter, compared with 30 and 34 micrograms per cubic meter for those reporting up to two hours per day and no workplace exposure respectively.

While the first of these studies suggests RSP increases due to smoking of less than a factor of three, the latter study shows virtually no increase in RSP exposures due to working with smokers.

In spite of these findings, the NPR concludes that the data cited are sufficient to support OSHA's risk assessment. OSHA summarizes the limited ETS exposure data presented in the NPR with statements regarding nicotine and ETS RSP and these are reproduced here in slide 18.

For nicotine, the NPR claims that the "ETS-nicotine exposures of the average worker appear to be of the order of 5 to 10 micrograms per cubic meter ..., and for the most exposed workers, [are] 50-100 micrograms per cubic meter."

"For EST-RSP, exposures are about tenfold that of the nicotine levels."

The summary conclusions drawn from the database assembled in the NPR are compared in my written submission to the largest, most representative, most recent and most relevant database on workplace exposures ever assembled, that is, the study by Oak Ridge National Laboratory.

Slide 19 compares the OSHA cited value for nicotine exposures with nicotine exposure levels actually measured by Oak Ridge. As seen, the NPR has overestimated average worker exposure levels 25 times and has overestimated most exposed worker exposure levels approximately 10 times.

Shown here in slide 20 is a comparison of the OSHA-cited value for ETS RSP with exposure levels actually measured recently by Oak Ridge.

As I said earlier, from the Oak Ridge study, ETS RSP can be determined for each workplace based on solanesol, the best available tobacco-specific indicator of the ETS contribution to RSP.

Based on these data, OSHA has overestimated average worker exposure levels approximately 250 times and has overestimated the most exposed worker exposure level approximately 15 times.

In summary, as demonstrated here, the NPR does not adequately address worker exposure to ETS and, as a result, OSHA has incorrect information regarding how much ETS American workers typically encounter in the workplace.

My fourth point in this section on ETS exposure addresses OSHA's comparison of workplace exposures for people who work with smokers to home exposures for people who live with smokers.

The NPR attempts to establish that ETS concentrations in these two environments are equivalent. An obvious question to ask is why would such a link be important?

The answer is quite simple. The attempt to equate exposures between home and workplace situations is prerequisite for attempting to substitute spousal smoking epidemiology for workplace exposure epidemiology.

Consistently and incorrectly the NPR asserts that ETS exposures are equivalent for living with and working with smokers. In this section of the NPR, OSHA appears to be arguing that ETS concentrations are equivalent for living with and working with smokers. Even if concentrations were equivalent in both venues, which as I will show momentarily they aren't, true exposures remains significantly different.

Slide 21 recalls the correct definition of exposure, that is, exposure equals concentration times time. It is straightforward to see that if the concentrations are equivalent in two environments, the ratio of exposures becomes equal to the ratio of the time spent in the two environments.

So the relevant issue now becomes how much time does a typical worker spend at work and at home? Don't forget to include days off, weekends, holidays and vacation days.

Most workers spend more waking hours at home than they do at work. Plus, they have sleeping time which hopefully also occurs at home. In a year, most people will spend far more time at home than they do at work. For example, a year consists of 8760 hours. The typical work year is only 2000 hours.

So how does this affect OSHA's proposition?

Well, even if you were to assume equal ETS concentrations at home and work, long-term exposures due to living with smokers will be far greater than long-term exposures due to working with smokers. However, that's a worst case scenario.

Let's now answer the question of how home ETS levels compare with workplace ETS levels, assuming of course there are smokers present in both places.

In this type of data analysis, it's not important to list the actual air concentrations or exposures in the home and workplace environments. What really matters, and it's a simpler way to look at the data, is the ratio between the two scenarios.

True exposure ratios between living and working with smokers are available from several recent population based exposure studies. These data have been collected by using either long-term personal monitoring, as was done in the survey we conducted in Ohio in 1991, or by using separate personal monitoring in the workplace and at home for one full 24-hour period, as was done in the survey we conducted in New Jersey in 1992 and in the recently completed study conducted by Oak Ridge

National Laboratory.

The relative weekly exposure estimates obtained in Columbus, Ohio can be calculated for three different ETS markers and are shown here in slide 22. These include the week-long personal monitoring for nicotine and 3-ethenylpyridine and also salivary cotinine measured four times during the one-week period.

The exposure ratios between non-smokers who were exposed only at home and non-smoker who were exposed only at work are 11 for 3-ethenylpyridine, 10 for nicotine and 6 for cotinine. Remember, these are exposure ratios. They indicate that non-smokers who worked with smokers experienced 6 to 11 times lower exposure than non-smokers who live with smokers. These data were submitted to OSHA in May 1993 and were published later that year.

Weekly estimate of home compared to work exposures from the New Jersey study are described in detail in my written submission. These data show average exposures are 2.9 times lower for working with smokers based on 30 different volatile organic compounds.

Additional data from that study showed 3.6 times lower average exposure for working with smokers based on five different particulate phase markers.

Based on three vapor phase ETS markers and four different indicators of the particulate phase of ETS, Jenkins et al. reached similar conclusions from the Oak Ridge study. Quoting from Jenkins in slide 23, "Although participants perceived their greatest exposures to ETS to occur in the workplace, in fact exposure to ETS when living with a smoker is about a factor of five greater than that received in a smoking workplace."

As summarized in slide 24, all of these data I have just presented which in total were gathered over the last three years in 14 different U.S. cities paint a very consistent picture of exposure due to working with smokers versus living with smokers. Using over 30 different markers of ETS exposure, these studies show that workplace exposures due to working with smokers are three to 11 times lower than home exposures due to living with smokers.

Taken together, these data indicate that true exposure to ETS between living with smokers and working with smokers is minimally three times lower and is most likely five to 10 times lower in the workplace.

Although these two studies were not cited by OSHA, let's move on to studies that were included in the proposed rule.

In two of the predominant studies cited in the NPR, the researchers asked subjects to guess how much ETS they were exposed to. Then they attempted to verify exposures by measuring cotinine. However, the NPR does not cite the cotinine data from either study. A proper consideration of these data actually refutes OSHA's claim regarding the magnitude of workplace exposure relative to home exposure.

Let's now consider what these two studies cited by OSHA actually show.

In the study by Cummings et al. and the data re-analysis requested by OSHA, 77 percent of workers reported ETS exposure at work. In the NPR, OSHA states, "This further analysis indicates that the workplace is a significant source of ETS exposure for non-smoking employed people."

Please recall the correct definition of exposure and realize that in the re-analysis there is no consideration for duration of exposure or ETS concentration. This reveals that the assertion put forth in the NPR is incorrect regarding what the Cummings et al. study actually shows.

Simply based on the percentage estimates, the only valid conclusion is that the workplace can be a significant source of potential exposure among the studied population.

To begin to evaluate objectively whether the workplace is a significant source of true exposure, an analytical measurement of exposure is needed.

In the re-analysis of the Cummings data requested by OSHA, urinary cotinine concentrations are provided according to the four exposure scenarios of all combinations of exposure at home and work. These data are reproduced in slide 25.

Clearly the effect of home exposure on cotinine levels is predominant. In fact, people reporting exposure to ETS only at work and not at home had the lowest cotinine levels of any group in the study, even lower than people reporting no exposure either at home or at work.

Cummings provides a peculiar explanation in an attempt to minimize the results of this re-analysis. This is slide 26. He states, "This analysis of cotinine values suggests that home exposure is more important than work exposure. However, this result is misleading since many of the subjects included in our study took time off from work to attend our clinic. Thus, cotinine values would, of course, be influenced more by home and public location exposures, not workplace exposure."

Such an explanation is hardly plausible, since cotinine has a fairly long half-life of about 17 hours and presumably most subjects would have been away from work for only one to two hours before providing the urine sample.

Assuming a simple model of first-order decay with a cotinine half-life of 17 hours shows that cotinine concentrations would decrease less than 8 percent after two hours away from the source of ETS exposure. Moreover, any decrease in cotinine concentrations incurred by coming to the clinic is equally applicable to any source of ETS exposure, whether it is at home, at work or in public places.

Slide 27 contrasts two very different views of what this study actually shows. The NPR states that the data of Cummings et al "present results to show significant workplace exposures to ETS."

In fact, Cummings' re-analysis of the cotinine data shows one thing quite clearly. That is, and this is very important, essentially there is no discernable effect of workplace exposure.

As additional evidence, I would like to quote from the second study cited in the NPR, that of Emmons et al. This passage on slide 28, however, was not cited in the NPR. "When those with and those without a smoker in the household were examined separately, we found that subjects who lived with a smoker received more exposure in the home than in the workplace."

In further describing the study of Emmons et al., OSHA claims that this study substantiates the magnitude of workplace exposures. The NPR also states, "For example, Emmons et al. found that the majority of ETS exposure occurred in the workplace." As I said before, OSHA appears to be using an incorrect definition of exposure.

As a consequence, the NPR reaches an inappropriate conclusion. What Emmons et al. actually show, like CAPS and like Cummings et al. is that the reported duration of ETS exposure in the workplace is higher than in the home.

Remember your guess as to how far I drove yesterday in an hour and a half?

So what does a proper consideration of the Emmons et al. study show?

Well, it actually raises two important issues. The first important issue is outlined here on slide 29. Emmons et al. report two components of potential exposure. That is, exposure duration in minutes per day and exposure intensity rated as near versus far. While the subjects rated approximately three times the potential exposure duration at work than at home, their ranking of intensity was approximately two times greater in the home.

Clearly true exposure is a combination of both duration and intensity or concentration. It is impossible to tell from the subjective data the net effect of these two differences.

Second, Emmons et al. did make an analytical measure of ETS exposure. However, this is not mentioned in the NPR. In a subsequent report on the same study, Emmons et al. give cotinine data.

Slide 30 reveals the original authors' conclusions regarding their own study. According to the authors, and again I quote, "Volunteers who lived with but did not work smokers had significantly higher cotinine concentrations than volunteers who were exposed to smokers only in the workplace. In addition, volunteers who had regular ETS exposure at home and at work had significantly higher cotinine ... than volunteers whose primary exposure was at work."

Regarding the same data set in another report, Emmons et al. state, "Subjects who lived with a smoker received more exposure in the home than in the workplace."

The cotinine results from Emmons et al. are reproduced in my written submission, along with the results from Cummings et al. and the more recent studies I described earlier.

The similarity of cotinine pattern among these five studies is striking. As emphasized on slide 31, the overwhelming conclusion from these data is this: ETS exposure at home for those living with smokers is a substantially larger contributor to elevated cotinine levels than ETS exposures at work for those working with smokers.

What happens when these data are compiled with new data from nationally representative monitoring studies that rely on multiple markers, including better markers, of ETS exposure?

The resulting data set portrays a very consistent pattern of ETS exposure that demonstrates working with smokers results in five to 10 times less ETS exposure than does living with smokers. This conclusion differs substantially from the position as stated in the NPR that exposures in the two venues are equivalent.

OSHA also states in the NPR the database on nicotine concentrations shows significantly higher average exposures in workplaces than in residences.

Before moving on to a discussion of what the nicotine data show, it's appropriate to pause for just a

moment and remember why so much attention is being focused on comparing home exposures with workplace exposures.

Quoting from the NPR on slide 32, "Thus risk estimates based on residential exposures are expected to accurately reflect occupational risks in most workplaces and possibly underestimate the risks in some workplaces."

Dr. Sears will address this topic further in his presentation.

Again, more recent, more relevant and more representative data which used nicotine concentration as an index of exposure in fact showed just the opposite.

Slide 33 reminds us that in the Oak Ridge study Jenkins et al. report nicotine exposures for smoke exposure away from work and smoke exposure at work venues. The ratio of the medians shows differential exposure to be a factor of 6.8 higher away from work.

This compares to the exposure ratio of 10.3 based on nicotine monitoring previously reported to OSHA from the study we conducted in Columbus, Ohio.

Thus, using an additional marker, nicotine, as the index of ETS exposure shows similar findings.

Working with smokers results in seven to 10 times lower exposure than living with smokers.

I would like to turn now to my final point on exposure and that is how many workers are actually exposed to ETS in the workplace?

Slide 34 shows that to estimate the prevalence of ETS exposure among the U.S. non-smoking workforce in the NPR OSHA attempts to discredit the largest and most representative survey done to date, that is, the NHIS survey. Instead, the NPR places far too much significance on a much smaller and clearly inappropriate study.

Proper consideration of these data reveals that OSHA has actually used the overall best estimate of population exposure as the lower bound in the risk assessment and suggests that the upper bound used in the NPR is biased high by at least a factor of two.

OSHA has not established a lower bound for the risk assessment. Let me briefly explain why this is true.

The NHIS, or the National Health Interview Survey, was conducted by the Centers for Disease Control and is the largest and most representative survey of its type done to date.

This survey found that 18.81 percent of the American workforce reports ETS exposure at work. The NPR states that this 18.81 percent "may be an underestimate of frequency of exposure in the workplace because it is based solely on self-reported information and the question was not very specific in defining immediate work area."

On its face, the information from NHIS would appear to be the definitive database for OSHA to use in characterizing the prevalence of occupational exposure to ETS.

On slide 35, let's look at the advantage and disadvantages of this study and try to figure out what, if

anything, is wrong with it.

Obvious advantages of NHIS include:

(1) It's a very representative study. In fact, it appears to be the exact population OSHA is trying to characterize.

(2) It is a very large survey, with over 7000 non-smoking respondents.

(3) A very recent survey conducted in 1991.

(4) It is the same survey used in the NPR to estimate the percentage of non-smoking workers in the U.S. that would be covered by this proposed standard.

OSHA levies two criticisms against the survey. The first criticism is that NHIS is based solely on self-reported information. How does one expect to find out how many workers in the U.S. are potentially ETS exposed if not by self-report? This has to be the starting point for obtaining the necessary information.

As is obvious from a careful scrutiny of the other two data sets OSHA has relied predominantly upon, that is, the studies of Cummings et al. and Emmons et al., which I have already described briefly, self-reported exposures actually tend to overestimate true exposures. On this first point, the NHIS study results would seem to be biased high, if anything, rather than low.

The second criticism in the Notice of Proposed Rulemaking is that the NHIS study was not very specific in defining immediate work area. The survey asked participants whether during the past two weeks anyone had smoked in their immediate work area. It would appear that this definition of exposure would actually include a significant number of respondents who are only incidentally or infrequently exposed.

Based on the question asked, exposure due to someone walking through one respondent's work area with a lit cigarette once during a two-week period gets the same weighting in this survey as the exposure for another respondent who might share an enclosed office with a smoker every day.

Also, it seems likely that most workers have a reasonably good concept of their immediate work area. To OSHA's second criticism, the NHIS study results would again seem to be biased high, if anything, and not low.

It appears that OSHA has exactly what it needs in this single study, a midpoint estimate of the prevalence of occupational exposure.

After evaluation of its strengths and weaknesses, this still appears to be the definitive database for OSHA to use in characterizing the prevalence of occupational exposure to ETS.

Adding further support to the prevalence of exposure information in this study is the similar prevalence of ETS exposure among the non-smoking workforce that we found in two studies which we conducted in 1992.

In one, a nationwide, random digit dialing telephone survey of non-smoking females, we found that 16 percent of working females reported exposure to ETS at work.

In the second, a mall intercept study, which was conducted in nine U.S. cities and included collection of saliva for cotinine determination, we found that 18 percent of confirmed non-smoking females reported exposure to ETS from their co-workers in their immediate work area.

These data, in combination with the NHIS data, demonstrate that approximately 16 to 19 percent of the U.S. workforce appear to be ETS exposed in their place of work.

Another source considered in the NPR for defining non-smoker ETS exposure prevalence in the workplace is the work published by Cummings et al. As shown on slide 36, OSHA states, "A recent re-analysis of the data file showed that among the non-smoking, currently employed subjects, 48.67 percent ... reported exposure to ETS at work and not at home." This is the number that OSHA uses as the estimate of the upper limit of the number of non-smokers exposed to ETS at work.

Let's look at the advantages and disadvantages of this study on slide 37. The only obvious advantage of this study is that it employed one analytical estimator of ETS exposure. That is, cotinine.

The obvious disadvantages of the Cummings et al. study are several, some of them are major. They include the following:

(1) It is a very non-representative study, deriving all study participants from a cancer screening clinic in Buffalo, New York by inviting them to participate in a study on ETS.

(2) It is a relatively small study, incorporated 339 participants.

(3) It is a relatively old study, conducted in 1986.

(4) According to Cummings et al. it "over represented females and whites and under represented persons below 40 years of age."

(5) It also was not very specific in defining exposure.

This is clearly a non-representative study, both in the selection criteria used for contacting potential subjects in a cancer clinic and the way subjects were invited to participate, by informing them it was an ETS study.

OSHA selects from the 78 percent who reported ETS exposure the 48.67 percent who reportedly were exposed at work and not at home.

The mean urinary cotinine level determined for the 48.67 percent of the study group is actually the lowest of all the groups, even lower than the group reporting no exposure either at home or at work.

So in essence, as I've outlined here on slide 38, OSHA uses as the upper limit for prevalence of occupational ETS exposure a number that is obtained from a group of study participants that on average had no detectable ETS exposure above background.

The NPR also refers to Emmons et al. as justification for the upper limit on the number of workers exposed.

OSHA states, and I quote, "Seventy-six percent of the subjects reported being regularly exposed to

ETS in the workplace. The percentage of subjects reporting exposure at work is similar to that found by Cummings et al."

The issue of study representativeness, that is, being representative of the U.S. population at large, is critical in a determination of the maximum number of potentially exposed non-smokers in the U.S. workforce. It is true that the percentages of the study populations reportedly exposed at work are similar between Cummings, 78 percent, and Emmons, 76 percent. However, the self-selective nature of recruited subjects was also similar in both studies.

Cummings et al. invited attendees at a cancer screening clinic to "participate in a study on ETS." Likewise, the subjects in Emmons et al. "were volunteers who responded to advertisements for a study on passive smoking." Also, the Emmons et al. subjects were selected to yield a wide range of ETS exposures, not necessarily to be representative. In fact, 106 of the 186 subjects represent just nine different work sites.

In no way can the reported prevalence or magnitude of ETS exposure among these study participants be generalized to the entire non-smoking U.S. workforce.

Given the nature of recruitment in both studies, it is surprising that even larger percentages did not report ETS exposure.

Think about this. Why would people volunteer to participate in a study on driving habits if the didn't drive?

Now think about this. Why would people volunteer to participate in a study on ETS exposure if they did not believe they were ETS exposed?

The fact that these two studies are not representative is no secret. In their paper, Cummings et al. plainly state that, "Given the self-selected nature of the study population and potentially limited generalizability of results ...."

Likewise, Emmons et al. describe their study population as "a motivated sample of convenience"

Again, proper consideration of these data reveals that the NPR has actually used the overall best estimate of population exposure as the lower bound in the risk assessment and suggests that the upper bound used by OSHA, that is, the 48 percent, is too high by at least a factor of two.

By under-emphasizing the most representative study and over-emphasizing an inappropriate study, the NPR has not established an accurate representation of the prevalence of ETS exposure among the non-smoking U.S. workforce.

Slide 39 concludes that the NPR's assessment of the non-smoking working population's exposure to ETS while at work is in accurate. Reliance on outdated, non-representative, extreme and limited data sets has led to substantial overestimation of true ETS exposure in typical U.S. workplaces.

As a result, OSHA concludes that non-smoker exposures due to living with smokers are equivalent to non-smoker exposures due to working with smokers.

True differences in exposure between these two populations are shown to be three to 10 times lower for occupational exposures in more recent studies that have been submitted to the docket.

In the proposed rule, the absolute magnitude of workplace ETS levels has been inferred from an extremely limited and biased data set derived mostly from short-term, peak concentrations measured by area monitoring over a decade ago.

Typical workplace levels appear to be 25 to 250 times lower than assumed by OSHA. By simply relying on the studies cited and data sets in the NPR, OSHA has significantly overestimated both the magnitude and prevalence of the workforce population's exposure to ETS.

In short, OSHA's proposed workplace ETS rule is based on misinformation. OSHA has not measured ETS exposure, OSHA doesn't know how much ETS is in the typical workplace and OSHA doesn't know how man workers are exposed to ETS at work. Therefore, OSHA's proposed workplace ETS regulations are unjustified.

Thank you.

JUDGE VITTONE: Thank you, Dr. Ogden.

Dr. Ogden's printed statement will be identified for the record as Exhibit 228. His slides will be identified as Exhibit 229.

(The documents referred to were marked for identification as Exhibit Nos. 228 & 229 and were received in evidence.)

MS. SHERMAN: Your Honor, would this be a good time for break?

JUDGE VITTONE: We'll take a five minute recess. And then we'll come right back. Please. I'm going to ask you to be precise on the time.

Off the record.

(Whereupon, a brief recess was taken.)

JUDGE VITTONE: Dr. Coggins, would you introduce the next speaker, please?

DR. COGGINS: Thank you, Judge.

Now Dr. Nelson will talk about the limitations in using nicotine and cotinine to precisely predict ETS exposure.

Dr. Nelson.

JUDGE VITTONE: All right. Dr. Nelson.

DR. NELSON: Thank you.

Good morning. I am Dr. Paul Nelson. I'm an analytical chemist with R.J. Reynolds tobacco company. For the past seven years I've been studying the chemistry and fate of environmental tobacco smoke, or ETS.

Specifically, my research has focused on three areas: measuring a quantity and behavior of ETS constituents; testing methods for ETS reduction and removal from indoor spaces; and third, helping to determine human responses to ETS.

(Slide Presentation)

Today I will discuss some problems with the use of nicotine as a quantitative marker for ETS levels and the use of nicotine and cotinine as quantitative biomarkers for ETS exposure.

In the proposed rule, OSHA claims that nicotine and its metabolized cotinine are good quantitative biomarkers for ETS. OSHA states that "nicotine and cotinine accurately predict the quantity of ETS exposure that has taken place over a recent time span." This claim is not justified by the existing body of evidence.

Why? There are two basic problems with giving nicotine and cotinine as quantitative biomarkers for ETS exposure.

First, nicotine does a poor job of predicting exposure to ETS constituents in the field, or "real world." Second, nicotine and cotinine concentrations in biological fluids do not accurately quantify past exposures to ETS. That is to say, the amount of nicotine and cotinine in your body right now cannot tell me when you were exposed to ETS, where you were exposed, or how much ETS exposure you had. As a result, nicotine and cotinine cannot be used to quantify ETS exposure. And they cannot provide a reliable basis for quantitative risk assessment.

Nonetheless, nicotine and cotinine concentrations can provide useful information in many circumstances. Especially in distinguishing between smokers and non-smokers.

Slide 2 illustrates the fact that nicotine exposure of smokers is much greater than the nicotine exposure of non-smokers. In general, regular smokers of more than a few cigarettes per day are easily distinguished from even the most heavily exposed non-smokers on the basis of nicotine or cotinine measurement in plasma, urine or saliva.

In addition, when looking at groups of individuals, cotinine measurements can distinguish groups who are not exposed to ETS from groups who are moderately or heavily exposed.

Nicotine is one of the easiest markers to measure in the field. It is often used to characterize the amount of cigarette smoke in the air. In conjunction with data from other ETS markers nicotine data can help provide a general indication of ETS levels. Furthermore, in controlled exposure studies nicotine and cotinine in body fluids can be used to determine the uptake of nicotine from the air. However, they do not necessarily indicate uptake of any other ETS constituents.

Before I demonstrate why nicotine and cotinine cannot be used as quantitative markers, or biomarkers, for ETS exposure, I should define what I mean by quantitative. For a quantitative relationship to exist, a numerical relationship must exist. In the case of ETS, a quantitative marker should predict the concentrations of other ETS constituents, both accurately -- that is, give the right number -- and precisely, which means reproducibly.

For example, the odometer in a car provides a quantitative indication of the number of miles traveled, regardless of the automobile used or the route followed. Terms such as "a lot," "far," or "not much" mean different things to different people and cannot be numerically related to the distance a car has

travelled. In a similar fashion, a number of terms frequently used to assess ETS exposure, such as "some," "a little," and "a lot" have no numerical basis and therefore cannot be used to quantify ETS exposure..

So why can't we use nicotine and cotinine to quantify ETS exposure? First, I'll talk about the use of nicotine for quantifying atmospheric levels of ETS. Then I'll discuss the problems with using nicotine and cotinine as quantitative biomarkers.

Nicotine fails to satisfy commonly accepted criteria for an ETS exposure marker. These criteria, which have been listed by the National Research Council, are shown on Slide 3. And they simply follow common sense.

First, the marker should be specific to ETS. Other sources of the marker should not be strong enough to substantially affect the marker concentration.

Second, the marker should be easy to detect.

Third, all cigarettes should have similar amounts of the marker.

And, finally, the ratio between the marker and the compound of interest should be constant, regardless of the brand smoked, the sampling location, ventilation rates or furnishings of an indoor space.

The first criteria, that a marker be unique to ETS is probably met in areas where smoking usually takes place. However, nicotine has reversible adsorption properties. That is, after ETS generation, nicotine easily sticks to surfaces such as walls, furniture and clothing. It is later re-emitted from these surfaces in the absence of other ETS constituents. So in areas where smoking takes place only occasionally, nicotine can sometimes be detected in the absence of other ETS constituents.

For example, let's say I had a party at my house last night where several people smoked. Even though no one is smoking there now, there's a good chance I'll be able to detect nicotine in the air today. So I could be exposed to nicotine without being exposed to other ETS components.

This characteristic appears unique to nicotine because no other ETS constituent is known to exhibit the same degree of adsorption or re-emission over a long time period.

The presence of nicotine in the absence of ETS has been demonstrated by a number of studies, including chamber and field studies where unexpected background nicotine measurements have been recorded in the absence of smoking. In addition, many researchers have demonstrated that nicotine decays or disappears from the air more rapidly than other ETS constituents. This provides further evidence of nicotine adsorption.

Because of this, nicotine and cotinine concentration could be found in biological fluids of individuals without their being exposed to ETS. For people with no or low ETS exposure, the detection of nicotine or cotinine in the body could be improperly ascribed to ETS exposure.

Now, let's look at the second NRC criterion, that the markers should be easy to detect even at low smoking rates. Does nicotine meet this criteria? Certainly. In fact, one of the main reasons nicotine has been measured so often is its ease of detection.

The third criterion, that cigarettes emit similar amounts of nicotine, is demonstrably untrue. There are

currently over 1,300 different brand styles of cigarettes on the U.S. market. Different styles, the same brand of cigarettes -- for example, Winston Box, Winston 100s or Winston Lights -- don't necessarily contain the same blend of tobacco or have the same combustion characteristics. In other words, they may generate different amounts of ETS.

As you can see here on Slide 7, nicotine yields of the top 50 U.S. brand styles show wide variation. An investigator testing a specific brand style of cigarette cannot simply assume that the cigarette tested represents the entire brand family. Likewise, an investigator who tested a small segment of the market, say 10 different cigarettes, cannot claim that such a sample is representative of the entire market.

The fourth criterion listed by the NRC, that there should be a consistent ratio between the marker and the contaminant of interest, is probably the most important criteria because it establishes the link between the marker and the agent of concern. This criterion has three elements.

First, there must be a contaminant or contaminants of interest.

Second, the ratio between the marker and the contaminant of interest must be consistent across a range of tobacco products.

And third, the ratio between the marker and the contaminant of interest must be consistent across a range of commonly encountered environmental conditions.

For many reasons, the use of nicotine as a marker for ETS fails to meet this criterion.

The first problem is this: If you don't know what you're looking for, how can you possibly choose a marker for it?

OSHA tends to deal with ETS as if all of its components behaved in exactly the same way. But they don't. Approximately 100 compounds have been identified in environmental tobacco smoke. Experimental data indicate that each of these constituents may potentially behave differently when introduced into indoor spaces. And nicotine's unique decay characteristics make it unsuitable as a marker for most identified constituents of ETS.

It's generally thought that if there are any health problems associated with ETS they might be related to compounds in the particulate phase. Like ETS in general, ETS RSP, or respirable suspended particles from ETS, is a complex mixture. Very little is understood about its composition. Even less is known about the relationships among ETS RSP constituents.

In the proposed rule OSHA has suggested that nicotine is a quantitative marker for ETS exposure. Therefore it must also be quantitatively related to ETS RSP exposure. But nicotine, a vapor phase component of ETS, is not a good marker for ETS RSP.

Why? First, nicotine and RSP yields of cigarettes are poorly correlated. Recently, Reynolds Tobacco conducted a study of the top 50 cigarette brand styles -- and this was the largest number of brands surveyed to date, accounting for 64 percent of the U.S. market.

As you can see here on Slide 9, emission rates for nicotine vary considerably. The sales-weighted average nicotine yield was about 1.6 milligrams per cigarette. But even near that average, as you can see here on this chart, RSP yields vary from below 10 to up close to 20 milligrams per cigarette. The nicotine yields of individual brands vary between 1 and 2.3 milligrams per cigarette. A regression of

these data indicates that nicotine and RSP yields are only poorly correlated, an "r" value of .47. And that's "r" as opposed to "r"-squared. And the relationship between ETS RSP yield and nicotine yield cannot be expressed as a simple ratio.

A second problem with using nicotine as a marker for ETS is that nicotine has not been demonstrated to be found in a consistent ratio to ETS RSP across a range of environmental conditions typically encountered. Studies at our laboratory have shown that the relationship between nicotine and ETS RSP can be consistent in a controlled environment test chamber operated at a single ventilation rate. However, as the data I have provided to OSHA previously show, that relationship breaks down at different ventilation rates or when measurements are taken in the field.

In a single environment, such as an office or a restaurant, one might expect that the relationship between nicotine and ETS RSP would remain constant.

Does it? Sometimes, yes. But often not.

During her testimony at this hearing, Dr. Katherine Hammond discussed the study she and Dr. Brian Leaderer conducted that purports to demonstrate that nicotine is a good predictor for RSP concentrations. These study has two main failings as applied to OSHA's proposed rule.

First, it was performed in homes, not workplaces. Ventilation rates in homes are generally low. Less than one air change per hour. Ventilation rates in workplaces are typically higher. This is important because the ventilation rate is a key factor associated with the ratio between concentrations of nicotine and ETS RSP.

Second, nicotine concentrations measured in homes do not appear to be highly predictive of RSP concentrations in this same environment. This is especially true at low nicotine concentration, which happen to be those most often encountered.

In recent years our company has conducted studies in several buildings owned and operated by Reynolds Tobacco. The methods and results of those studies have been submitted to the docket. I would like to discuss those results that pertain to the use of nicotine to predict exposure to ETS RSP.

In one study, RSP and nicotine were measured in several locations on a single floor of a large office building. The relationship between RSP and nicotine for the different sampling locations is presented on Slide 10. RSP and nicotine were not significantly correlated at any of the five sampling locations. That is, they were independent of each other at every location.

Slide 11 incorporates the data for all sampling locations. Looking at the overall data, there was no statistically significant correlation between nicotine and RSP.

We also looked at the relationship between nicotine and ultraviolet particulate matter, or UVPM. UVPM is more specific that total RSP as a marker for ETS RSP. In discussing field measurements, I will use UVPM as a surrogate for ETS RSP.

Slide 12 shows the relationship between nicotine and UVPM for each of the sampling locations. UVPM was statistically significantly correlated with nicotine in only two of the five sampling locations. Where correlations were statistically significant, they were also poor -- "r" values were .63 and .64 for the two locations.

Slide 13 incorporates all the data points from all locations. The overall correlation between nicotine and UVPM was statistically significant. But the precision of the prediction was poor. So significant errors in accuracy could occur if nicotine were used to predict ETS RSP concentrations in the office building.

Why is that? Well, let me give you an example.

If I were in this building -- and it's the one illustrated on the slide -- and I were exposed to 2 micrograms per cubic meter of nicotine, the corresponding amount of ETS RSP I was exposed to could have ranged from as low as 5 to as high as 25 micrograms per cubic meter, a five-fold difference.

Similar results were obtained in a study of an office area in a different building. The existence of data sets that show a correlations between nicotine and RSP do not provide sufficient justification for assuming that nicotine is found in constant proportion to other ETS constituents within or across environments.

To put it very simply, if you see a couple of red cars on the road, you can't assume that all cars are red. Many data sets, including references cited in the proposed rule, demonstrate the nicotine is not found in constant proportion to RSP within single environments. It would be an error for OSHA to assume that the ratios between nicotine and other ETS constituents are consistent, or that nicotine is a good quantitative marker for ETS RSP.

Slide 14 shows the results of 480 concurrent area measurements of nicotine and RSP from workplaces in 15 U.S. cities. As you can see, the correlation between nicotine and RSP is not significant, and also very poor.

In an effort to compare Dr. Hammond's home data and the data derived in workplaces, I plotted the workplace data on the same axes that Dr. Hammond used in her submission to the docket. As you can see... And these are the axes: 0 to 20 micrograms per cubic meter nicotine and 0 to 200 micrograms per cubic meter RSP.

As you can see on this inset graph, the data are gathered in what appears to be a random pattern. In addition, many individual data points correspond to a wide variety of RSP and nicotine ratios. Because of the diversity in U.S. buildings in terms of the interior surface and ventilation characteristics, any correlation between RSP and nicotine derived from small sample sets or from samples collected in similar micro-environments, such as homes, cannot be applied across the range of environments encountered in the U.S. workplace.

During her testimony, Dr. Hammond suggested that variable background concentrations of RSP from sources other than ETS explain the variety of RSP-nicotine ratios observed in the field, and especially the variation and low nicotine concentration. It is true that non-ETS RSP can lead to unusually large ratios in some circumstances. However, it is unlikely that background RSP can explain all the variations in RSP to nicotine ratio.

The two data sets I'm about to describe illustrate this point. Back here on Slide 14, and I'm referring now to the inset on Slide 14, at nicotine concentrations of 4 micrograms per cubic meter there's a series of data points where the RSP concentrations are between 150 and 200 micrograms per cubic meter. The RSP to nicotine ratio is large, approximately 35 to 1.

Using Dr. Hammond's model, which is based on a ratio of approximately 11 to 1, one would expect ETS RSP to approximately 43 micrograms per cubic meter. That would require an ambient background in excess of 100 micrograms per cubic meter. A concentration much greater than Mr. Hammond testified that she would typically expect.

Obviously, many data points on this graph demonstrate the possibility of RSP-nicotine ratios greater than 11 that are unlikely to be explained solely on the basis of high background levels of RSP. Likewise, many data points correspond to a ratio much less than 11.

In the group of points between 7 and 8 micrograms per cubic meter, there are several data points where RSP concentration is less than 50 micrograms per cubic meter. For these points, the RSP-nicotine ratio would average about 3 to 1.

There is no way to explain this low ratio on the basis of background RSP. Dr. Hammond has correctly observed that background RSP can play havoc with RSP-nicotine ratios. And it can have a large influence. But it is not the sole reason that RSP-nicotine ratios are variable. And it may contribute only a small amount to the variation of ratios seen across a wide range of environments.

What other factors are important? Ventilation patterns, and by that I mean air distribution, as well as ventilation rates, or the amount of air you put into a space, and interior surface characteristics can also play a large ratio in the inconsistent relationship between ETS RSP and nicotine.

Now let's look at a second data set that illustrates that background RSP is not solely responsible for the wide variability in RSP-nicotine ratios.

As I noted earlier, ultraviolet particulate matter, or UVPM, is a marker that is more specific to ETS RSP because it does not measure most non-ETS particles. Consequently, the ratio of UVPM to nicotine should be relatively constant if, as Dr. Hammond suggests, non-ETS particles are the sole reason for the variation in RSP-nicotine ratio.

As the data on Slide 15 show, the ratio between UVPM and nicotine is not constant. We have a number of cities and locations, offices and restaurants -- I'm just explaining the chart. These are the average ratios, and that's the ratios of the measurements determining those individual, from individual measurements within these locations. Some standard deviation, minimum and maximum measurements observed. Anyway.

For example, in Winston-Salem offices, the average UVPM to nicotine ratio was 21.3, but the ratios range from as little as 1.3 to as high as 211. And the average ratio determined in the Winston-Salem offices is different from that determined in Dallas offices. These variations in ratio between ETS RSP and nicotine could not occur unless factors other than just background RSP are responsible for the variation in RSP to nicotine ratios.

So what do these results mean?

First, nicotine cannot be used to quantitatively predict concentrations of either RSP or ETS RSP in the indoor environments. These types of predictions are not accurate because the ratio of nicotine to RSP and ETS RSP is highly variable and dependent upon many environmental factors.

Second, no data demonstrate that the ratio of nicotine to any other ETS constituent is constant across

the wide range of the indoor environments encountered in the U.S. workplace.

Now that we've explored some of the problems with using nicotine as a quantitative ETS marker, let's look at the problems in using nicotine and cotinine as quantitative ETS biomarkers.

As we've seen, the relationship between exposure to nicotine and other ETS constituents is poor and highly variable. As a result, the prediction of exposure to individual ETS constituents on the basis of measurements of nicotine and cotinine in biological fluids must also be poor.

Some of the most significant problems was relating nicotine and cotinine concentrations in biological fluids to nicotine exposure include -- and they're here on Slide 16 -- a lack of standardized measurement techniques, inter-individual variation in the metabolism and clearance of nicotine and its metabolites confounding in some individuals by exposure to nicotine from sources other than ETS and the time dependent function describing nicotine metabolism in the body.

To the first point. Since standardized analytical methods don't exist -- and that's for measuring nicotine and cotinine in body fluids -- the results of nicotine or cotinine determinations obtained in one laboratory cannot necessarily be compared to those obtained at another laboratory.

A study performed by Dr. Jeffery Idle showed that there were considerable differences in reported cotinine concentrations from identical urine samples tested at different laboratories. These results are described in more detail in a previous submission to OSHA.

Point two. There are significant inter-individual differences in metabolism and clearance rates and to the relative proportion of metabolites generated. Exposure to a single concentration of nicotine can result in a wide range of nicotine or cotinine concentrations in different individuals.

As an example. If Drs. Coggins, Sears and I had a 10 microgram nicotine uptake right now, our salivary cotinine levels would almost certainly increase by different amounts. The differences can confound attempts to use spot nicotine or cotinine measurements to accurately and precisely back-calculated nicotine exposure.

The third problem, confounding by exposure to environmental nicotine in the absence of ETS or ingestion of nicotine from dietary sources has been discussed in detail in my previous submissions to OSHA.

Now I'd like to spend a couple of minutes exploring the fourth problem with using nicotine and cotinine as quantitative markers for ETS: The fact that concentrations of these compounds in biological fluid are highly time dependent.

At Reynolds Tobacco we have developed a physiologically-based pharmacokinetic, or PBPK model, that can predict general trends in concentrations of nicotine or its metabolites in the body fluids of smokers. We have applied this model to time dependent ETS exposure data.

Before I proceed, I should point out that PBPK models cannot predict nicotine or cotinine concentrations in any individual unless specific, relevant pharmacokinetic data are available and exposure information is know. Or multiple biological fluid samples have been obtained from that individual.

In this case, our model was used to predict plasma nicotine concentrations for an ideal individual. And by that I mean one for whom all the aforementioned information is available. We assumed an eight hour workplace exposure at 10 micrograms per cubic meter nicotine. We also assumed that the individual had no non-workplace nicotine exposure.

What does that profile look like?

Slide 17 shows the predicted plasma concentration of nicotine versus time. The data illustrate three important points that make it difficult to predict nicotine exposure on the basis of spot nicotine measurements.

First. The predicted serum concentrations of nicotine are extremely low.

Second. Nicotine concentrations in serum are extremely transient or variable. They vary throughout the course of the day. In fact a wide range of spot plasma, and therefore urinary and salivary, nicotine concentrations result from a single exposure.

Third. Nicotine is metabolized quickly. Within 16 hours following exposure -- the exposure ending here -- there is almost no nicotine particulate to be found in the plasma. Less than a day after exposure, the nicotine is essentially gone.

So what about using cotinine concentrations instead? There are similar problems.

As you can see here on Slide 18, cotinine concentrations also vary considerably across time. Because of this, at some time points, different nicotine exposures can result in identical spot cotinine concentrations. So that predicting ETS nicotine exposure based on cotinine or nicotine measurements in biological fluids is difficult to impossible, assuming the ideal conditions used in the PBPK model.

Where field measurements are concerned, the use of nicotine and cotinine as biomarkers to quantitatively predict ETS nicotine exposure are further complicated by inter-individual variability in nicotine and cotinine metabolism and inter-individual variability of physiological parameters related to adsorption, distribution and elimination of nicotine and/or its metabolites.

(3) Non-workplace exposure to ETS.

(4) Exposure to non-ETS nicotine.

(5) Much lower concentrations of ETS in the real world than used in the model and corresponding problems with working near the detection limit of analytical methods.

And, finally, poor inter-laboratory precision of nicotine and cotinine determinations.

Because of these factors, at low exposure levels nicotine and cotinine might not even serve as valid qualitative biomarkers for ETS exposure.

Even Dr. Neil Benowitz, one of OSHA's own witnesses at this hearing, has acknowledged many of these difficulties. A study conducted by Reynolds' scientists illustrates that the difficulties associated with predicting nicotine exposure on the basis of cotinine measurements.

Slide 20 shows the salivary cotinine measure in 20 subjects before and after ETS exposure. Throughout the week of the experiment the subjects were instructed to avoid ETS exposure in the evenings and at night. During days 1, 2, 4 and 5, the subjects stayed in an ETS-free environmental chamber throughout the day. On the third day of the experiment, the subjects were exposed to an average of 59 micrograms per cubic meter ETS nicotine for seven and half hours.

And I should point out that this is a typographical error on my written testimony and on the slide. This is seven and a half hour duration.

The ETS RSP concentration in the exposure chamber was 200 micrograms per cubic meter. Salivary cotinine was collected at the beginning and end of each day.

What were the results?

Well, looking at Slide 20, you can see that on average ETS nicotine exposure increased salivary cotinine. However, the important point here is that salivary cotinine concentrations cannot even be used to distinguish non-exposed individuals from those who have heavy ETS nicotine exposure.

As you can see, the salivary cotinine concentration of some individuals who had claimed they were not exposed to ETS were higher than the concentrations of some individuals who were exposed, or following the seven and a half exposure to ETS nicotine.

In addition, immediately following exposure small increases in salivary concentrations were detected in some individuals and substantial increases were noted for others.

Fifty-nine micrograms per cubic meter nicotine is much, much higher than anyone would normally expect to find in the vast majority of workplaces. As numerous scientists have recently testified, data obtained from field studies suggests that in the past average nicotine concentrations in smoking workplaces was approximately 10 micrograms per cubic meter and is considerably less in more modern workplaces.

Small increases in salivary cotinine following the experimental exposure would be considerably smaller upon exposure to ten, two or one microgram per cubic meter nicotine. It is likely that inter-individual variations in nicotine and cotinine metabolism and excretion would far outweigh the small, incremental increase in cotinine concentration following exposure to typical levels of ETS nicotine.

In other words, the variation between people is larger than the variation caused by normal exposures.

In any event, it is improper to suggest that nicotine, and hence ETS exposure, can be quantitatively determined on the basis of single, and perhaps even multiple, nicotine or cotinine measurements. If anyone who is interested in determining ETS exposures then they must first define specifically what exposure they are referring to and then measure that exposure directly. Or use a properly validated marker.

Now, if you say what do I mean by a properly validated marker, one that satisfies the NRC criteria. Especially the criteria that the marker be a consistent ratio to the component of interest, and also one that yields similar results regardless of the laboratory performing the analysis.

In conclusion, the information I have just presented only highlights some of the problems associated with the use of nicotine and cotinine as quantitative markers or biomarkers for ETS exposure. And

that brings me back around to my first slide and the real question here.

Are nicotine and cotinine quantitative ETS markers or biomarkers?

I think when all the information is considered the evidence strongly suggests that no quantitative relationship exists between ETS and nicotine and other ETS constituents. Spot measurements of nicotine and cotinine in body fluids cannot be used to predict ETS nicotine exposure. And nicotine and cotinine cannot be used to accurately quantify or differentiate among typical levels of exposure to other ETS constituents.

As a result, nicotine and cotinine cannot, and should not, be used to evaluate or predict risk associated with ETS exposure.

Thank you.

JUDGE VITTONE: Thank you, Dr. Nelson.

Dr. Nelson's printed remarks will be identified in the record as Exhibit 230, and the copy of his slides which he used in his presentation will be identified as Exhibit 231.

(The documents referred to were marked for identification as Exhibit Nos. 230 & 231 and were received in evidence.)

JUDGE VITTONE: Gentlemen, it's 12:00, and I propose that we break for lunch now.

Let me ask Dr. Coggins. Dr. Sears is next?

DR. COGGINS: Dr. Sears is next.

JUDGE VITTONE: Followed by whom?

DR. COGGINS: Dr. Ogden will come back, and then myself, then Mr. Bohanon. Four more speakers.

JUDGE VITTONE: Okay. One of those is a relatively short presentation, right?

DR. COGGINS: Mine's about 45 minutes. Ogden's is about 30 and Mr. Bohanon's is about an hour.

JUDGE VITTONE: About an hour. Okay.

MS. SHERMAN: Would it make sense to get one more before lunch?

JUDGE VITTONE: I was going to say, do you want to go one more time? Are you all ready?

Okay. Let's take Dr. Sears, and then we'll break for lunch.

(Pause)

JUDGE VITTONE: Dr. Sears, you may begin.

DR. SEARS: I'm Stephen Sears. I am a scientist in the research and development department of R.J.

Reynolds Tobacco Company. By training I'm a theoretical chemical physicist, and I have a Ph.D. in theoretical chemistry from the University of North Carolina at Chapel Hill.

For the past four years my work at Reynolds Tobacco has focused on environmental tobacco smoke and issues surrounding ETS risk assessment. Today I will talk about just some of the many problems with the epidemiologic approach the proposed rule uses to quantify risk. Specifically, I will explain why the epidemiological evidence that OSHA relied on does not support the contention that exposure to environmental tobacco smoke increases the risk of lung cancer or heart disease.

This discussion is detailed in the written critique that was prepared by Mr. Thomas Steichen and myself for submission to the OSHA docket. Mr. Steichen is a statistician and analyst in the R&D department of Reynolds Tobacco. He holds a Master's degree in statistics from the University of Kentucky and he is co-author of today's presentation. Mr. Steichen has been involved in ETS risk assessment issues for more than three years.

To reach its conclusions concerning ETS the proposed rule relies on the epidemiological evidence that has three key shortcomings, shown here in Slide 1.

First, the evidence is inappropriate for the workplace.

The second shortcoming. The evidence is methodologically flawed.

The third shortcoming. The evidence is insufficient to conclude increased risk.

There are also errors in the way this evidence was interpreted and applied. Therefore the conclusions drawn from this evidence are invalid.

During the past several years Mr. Steichen and I have extensively investigated the epidemiologic evidence concerning ETS exposure, lung cancer and heart disease. We have also analyzed how that evidence is used in the proposed rule. Based on that research there are a couple of points I'd like to make before I detail some of the problems in the proposed rule.

I show these points on Slide 2.

First, the epidemiologic evidence in general does not support the contention that ETS exposure increases the risk of lung cancer or heart disease. Among other flaws, the proposed rule relies on world-wide spousal exposure data to characterize U.S. workers. In fact, as you will soon hear, there are a number of good reasons for not using it. That is, there's a very strong argument for confining the evidence to data about U.S. citizens, and ultimately to data about U.S. workers.

Second. Even if the proposed rule had limited itself to U.S. spousal data, it still could not have reached the conclusion that ETS exposure increases the risk of lung cancer or heart disease.

For example, an EPA-style meta-analysis that includes the Brownson, Stockwell and 1994 Fontham data does not show an increased risk of lung cancer at the 95% level. Concerning ETS and heart disease, as everyone on the panel knows, the body of evidence is so inconclusive that the EPA abandoned its effort to deal with heart disease in its risk assessment.

And that evidence did not even include the results of three large studies that were recently introduced at these hearings. Those studies -- the American Cancer Society's two cancer prevention surveys and

the National Mortality Followback Survey -- have greatly increased the amount of data and the strength of the epidemiologic evidence concerning ETS and heart disease. They showed no statistically significant increased risk.

As an aside, the data from these three studies was available in 1972, 1988 and 1986, respectively. The fact that the results were never reported by the researchers highlights the substantial publication bias that exists with ETS.

Now. Let's look at why the epidemiologic evidence in the proposed rule in inappropriate for quantifying risk in the U.S. workplace.

The proposed rule uses world-wide spousal studies instead of U.S. workplace studies to evaluate risk to workers in the workplace. To justify this questionable approach, the proposed rule relied on three false assumptions. These were shown on Slide 3.

First, that spousal residential data is relevant to workplace risk.

Second, that workplace and residential ETS exposures are comparable.

And third, that the expected risk for workplace and residential exposures are equivalent.

The errors in these assumptions lead OSHA to the unjustified belief that world-wide spousal data are an adequate substitute for U.S. workplace date. This unjustified belief undermines the validity of the approach used in the proposed rule.

Let's look at each of the three assumptions in order. Slide 4.

First, are spousal data relevant to workplace risk? The studies cited in the proposed rule used smoking status of the spouse as a surrogate for ETS exposure. This conveniently let researchers label cases and controls as exposed and unexposed.

But these categories, in fact -- Slide 5 -- represent the cumulative and interactive effects of all the differences between the groups. That is, the full array of risk factors associated with living with a smoker. These factors include diet, exercise, socio-economic status, alcohol consumption and a host of others. So rather than being a surrogate for ETS exposure, living with a smoker is in fact a surrogate for the full range of independent risk factors that living with a smoker entails. As a result, risk ratios can only be computed for this aggregate of risk factors rather than for the ETS exposure factor alone.

The attendant statistical tests of significance do not and cannot indicate the specific risk factors that caused the two groups to appear significantly different. That is, the statistical test merely provides evidence that the two groups are or are not different. It does not provide evidence about why the groups are different.

In a November, 1994 publication, The American Journal of Epidemiology, Professor Sander Greenland noted that confidence intervals represent the minimum level of uncertainty associated with a conclusion. That is, the actual uncertaint